HAND BOOK
OF THE
SPHYGMOGRAPH.

Dr Burdon Sanderson. F.R.S.

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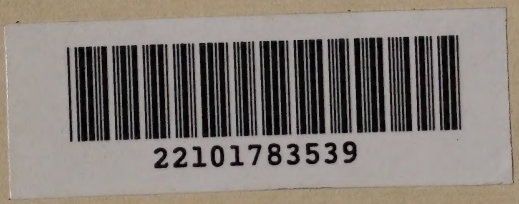
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THE

SPHYGMOGRAPH.

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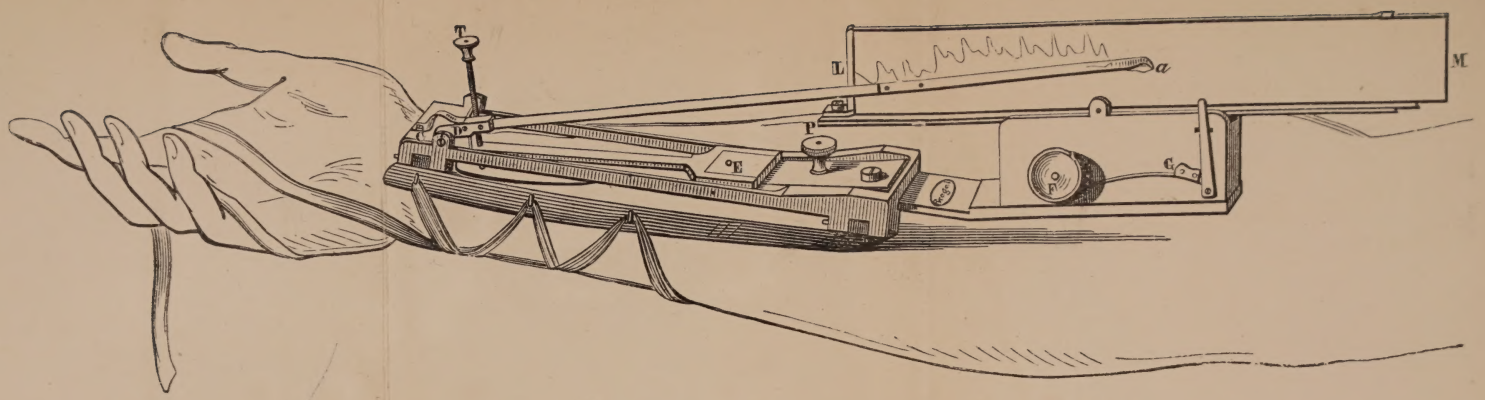

FIGURE 1.—The Sphygmograph applied to the Fore-arm.

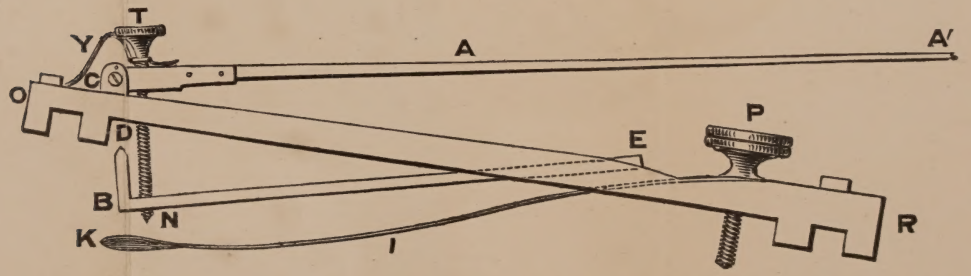

FIGURE 2.—Enlarged view of the frame of the Sphygmograph in profile, shewing the arrangement of the levers.

A A′ *a*, the writing lever, the axis of which is at C; K, the spring; B, the lever by which the movements of K are transmitted to A; D, its knife edge; T, an adjusting screw for varying the distance between K and D according to the pressure required; P, an adjusting screw for permanently adjusting the spring at the proper obliquity; F, G, the box in which the clockwork is contained; L, M, the traveller.

N.B. The same letters are used in both figures.

[To be inserted opposite the Title-page.

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HANDBOOK

OF

THE SPHYGMOGRAPH:

BEING A

GUIDE TO ITS USE IN CLINICAL RESEARCH.

TO WHICH IS APPENDED

A Lecture

DELIVERED AT THE ROYAL COLLEGE OF PHYSICIANS

ON THE 29TH OF MARCH 1867

ON THE

MODE AND DURATION OF THE CONTRACTION OF THE HEART IN HEALTH AND DISEASE.

BY

J. BURDON SANDERSON, M.D. F.R.S.

Physician to the Hospital for Consumption, Assistant Physician to Mid[illegible]ex Hos[illegible]al, and Joint Lecturer on Physiology in the Middlesex Hospital Medica[illegible]llege.

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PREFACE.

I OFFER this little book to my fellow-workers, in the hope that it may help them to overcome those preliminary difficulties which are apt to be encountered in the application of any new method of research, and that, in this way, it may tend to prevent the loss of time that might be more usefully employed in collecting and recording observations. Those who require only a grammar of the sphygmograph will, I trust, find what they want in the first two chapters. The more general questions relating to the physiological pathology of the circulation which are connected with the movements of the heart and arteries, are discussed in my Lecture, delivered at the College of Physicians last spring; while, in the third chapter, I have brought together certain clinical facts and

observations, which have little or nothing in common excepting their importance in relation to the pulse.

I have only to add, that my book makes no pretension to be regarded as a physiological treatise. I have designedly dealt with physiological theories in as cursory a manner as possible, making it not so much my aim to investigate them as to apply them to pathology. In doing so I have taken for granted some observations of my own not yet published, particularly those relating to the velocity of the transmission of vibrations, and to the production of successive movements, identical with those of the pulse, in elastic tubes containing liquids.

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HANDBOOK

OF

THE SPHYGMOGRAPH.

CHAPTER I.

THE SPHYGMOGRAPH.

Purpose and Construction of the Sphygmograph.

THE PULSATION felt by the finger, when placed on the radial artery at the wrist, is principally due to the fact that the artery enlarges during the contraction of the heart, and returns to its previous condition during the diastolic relaxation. Consequently, whenever a superficial artery rests, as the radial does, on a bony surface, every part of its subcutaneous surface moves up and down—just as, when a man is made to lie on a board on his back, the expansive movements of his chest are represented by up and down movements of the sternum. It is the main use of the sphygmograph to transfer these up and down movements to paper, in such a manner as to render it possible to measure their extent and relative duration. This measurement is not, however,

all that is required. If the investigation is to be complete, we must have a means of estimating the 'compressibility' of the artery, both during the contraction of the heart and during its repose. This problem is entirely apart from that of the measurement of expansive movement; and requires separate investigation.

If you place a long light slip of wood or ivory on the front of the forearm, in such a way that one end is supported on the scaphoid bone, while the other extends along the inner side of the radius, and if the wrist end of the slip be so fixed, or held in the position indicated, as to allow it to move up and down at its opposite end in the manner of a lever of the third order, having its fulcrum at the wrist, the arrangement will serve to show how the arterial movements may be so amplified as to admit of measurement. For at a point about an inch from its fixed end, the slip of wood will rest on the pulsating artery, and will acquire its motion. And supposing the lever to be 10 inches long, its free extremity will move through about a centre of an inch, for every 100th of an inch of linear expansion of the artery. If a sheet of paper, moving by clockwork at a uniform rate, could be so placed as to be marked by a pen suitably fixed at the free end of the lever above described, the whole would constitute a kind of sphygmograph. But, even if an

instrument were constructed on this principle in the most perfect manner possible, so that its axis of oscillation should work in bearings so fixed to the wrist as to be of one piece with the radius, it would be of little value. For, on the one hand, if the lever were made very slight, it would not keep in apposition to the artery; whereas, if it were made heavy enough to overcome this difficulty, the successive movements communicated to it would be modified by its own weight, particularly as regards their relative duration. For these reasons the notion, which at first sight appears so feasible, of transferring the arterial movements *directly* to paper, by means of a single lever, must be abandoned.

In the sphygmograph of Marey, the movements recorded are not those of the artery, but those of an elastic tongue of steel which presses upon it. This spring is screwed, at the end opposite to that which is applied to the artery, to a frame of brass, which is maintained in a fixed position as regards the radius, so that the pressure exerted by the spring is continuous and constant. It is manifest that, inasmuch as the spring depresses the surface of the artery, its movements are not identical with those of the arterial wall; hence the *extent* of motion is inaccurately measured. As, however, the *duration* of each motion can be determined with extreme precision by Marey's instrument, it must be regarded

as superior to any other which has been proposed, notwithstanding the defect above referred to.

The engravings opposite the title-page, borrowed from Marey's work,* will enable the reader to understand the general construction and mode of application of the sphygmograph. It is secured in its position by a bandage passed round the wrist, and laced alternately from side to side round little hooks, which are arranged along the edges of the metal wings, attached by hinged joints to the opposite sides of the instrument. In the interior of the framework, D R (fig. 2), seen in profile in the figure, is a flexible steel spring which descends obliquely, and is covered at its extremity by a plate of ivory, K. When the instrument is used, this plate rests upon the artery, which it depresses by virtue of the elasticity of the spring. This being the case, each pulsation communicates slight movements to the plate. To amplify these movements, a light wooden lever, A A′, of the third order, is used, which is supported by steel points at C. B E is a second lever of the same order, which has its centre of movement at E. It terminates at D, in a vertical knife edge, and is traversed by the vertical screw,

* 'Physiologie médicale de la Circulation du Sang.' Paris, 1863. For the stereotype blocks from which the engravings are printed, I am indebted to the kindness of Mr. Milne, of Upper Chadwell Street, the agent of M. Bréguet of Paris, the maker of Marey's sphygmograph.

T. When the extremity, N, of the screw rests upon the spring above the ivory plate, every movement of the plate is transmitted to B E, and by means of the knife edge to the wooden lever, A A′. The purpose of the screw, T, is to vary at will the distance between the wooden lever and the upper surface of the spring, without interfering with the mechanism by which the movement is transmitted. As the distance between C and D is much less than the length of the lever, the oscillations of its extremity, A′, are much more extensive than the vertical movements of the spring. The lever ends at *a* in fig. 1, in a pen of peculiar form, which writes on a slip of glazed paper supported on a metal frame. This frame moves by clockwork from M towards L, the rate of movement being such that about three inches are traversed in the tenth of a minute.

Defects and modifications.—The sphygmograph of M. Marey, as made by M. Bréguet, is not only a very perfect instrument as regards its plan of construction, but has the additional recommendation that it is exquisitely finished. It has, however, one or two defects, which are of so important a character, that, in procuring one, it is advisable to have it altered, before beginning to work with it. Fortunately, the necessary modifications can be very easily made. I will first refer to the way in which the instrument is secured in its place on the wrist. The mode of

adjustment ought clearly to be such as to enable the observer to regulate the position of the sphygmograph with the utmost facility, so as to allow of the ready application of the spring to the artery at the most favourable situation; it is equally essential that, when once properly fixed, the instrument should not stir from its position in relation to the radius, either laterally or vertically.

It was from the first evident, that, until some more ready and certain method of fixing the instrument could be contrived, it could not be made practically useful for clinical research; for, when the preliminary adjustment is attended with much loss of time and difficulty, both patient and physician are apt to become wearied long before any satisfactory result can be obtained.

M. Marey's method of fixing is defective, because the sphygmograph, instead of resting rigidly on the skeleton, is secured by bandages, which retain it steadily in its position only so long as the patient's muscles remain immovable. To remedy this, I tried in succession various mechanical contrivances, in all of which it was my endeavour to make the framework of the instrument as it were of one piece with the radius and ulna, which, near the wrist, may be regarded as one bone. Having failed to accomplish this, I had recourse to another expedient, which, although much simpler, I have found by experience to be efficient.

The plan I now adopt is based on the same principle as that which I had in view in my previous trials. It consists in adjusting to the end of the instrument (between the letters o and D in fig. 2) a rectangular block of brass, by the under surface of which it rests on the tendon of the *flexor longus pollicis*, and on the space between that tendon and the spine of the radius; the block being kept closely applied to the surface by means of a strong elastic band, which encircles the wrist. By this method the framework of the sphygmograph is made to rest firmly on a surface of bone, so that the axis of the lever is maintained at a constant distance from the artery. Its defect obviously lies in the fact, that the *point d'appui* is not the radius (the bone on which the artery lies) but virtually the *os scaphoides*, the relative position of which to the radius varies according as the wrist is flexed or extended. But this difficulty is entirely got rid of, if the forearm is so supported and controlled in its movements as to keep the hand bent backwards. For this purpose no expedient can be better adapted than the pad contrived by Mr. Berkeley Hill. This consists of a board 9 inches long and $3\frac{1}{2}$ inches broad, the upper surface of which is well padded and covered with stout leather. At one end the padding is of treble thickness, so as to form a cushion.

In use the pad is laid upon a table, the right arm

of the patient lying on the cushioned surface in the supine position, the back of the wrist resting on the thickest part, with the knuckles touching the table. This done, the hand is kept steady by means of strong elastic bands, which are attached to the opposite edges of the board. The sphygmograph is then applied in its proper place, and adjusted in the manner already described. When this plan is followed, it is obvious that although the sphygmograph rests principally on the tendon of the *flexor longus pollicis*, its relative position to the radius is quite as invariable as if it rested on the radius itself, so that the pressure exerted on the spring by the artery is constant,* so long as the distance between the wooden lever and the spring is unaltered.

It may be readily understood that it is of great practical importance to be able to vary the pressure, for it is by comparing the effects of different known pressures that the 'compressibility' of the pulse is to be judged of. With this object in view the sphygmograph is furnished with an adjusting screw, P, by turning which the inclination of the spring can be

* In order to fix the brass block described in the text to the sphygmograph, the japanned wings must be first removed, the pins by which these are attached being available for securing the block in its place. The thickness of the block, measured vertically, is an eighth of an inch, but it may be increased by sliding over its lower surface other pieces of brass, which are made of several thicknesses, so as to enable the observer to vary the distance between the frame of the sphygmograph and the artery, according to circumstances.

varied. If the block I have recommended be used, this mode of adjustment is not required; the screw, however, is still of great service, as affording the means of fixing the spring permanently in that position which is found practically best adapted for the investigation of pulses of ordinary firmness. For this permanent adjustment I have adopted the following method. Having found by experience, that it is not expedient to work with any less pressure on the artery than 100 grammes, and that the greatest distance between the wooden lever and the spring which the construction of the instrument admits of, is $\frac{11}{20}$ of an inch, it became my object to tighten the screw, P, to such a degree that the force required to bend the spring back to the distance indicated should be equal to the weight of 100 grammes. For this purpose I fix the sphygmograph in a vice, and by means of a counterpoised loop of wire attached to the end of the beam of a balance, the opposite pan of which contains a weight of 100 grammes, exert the required pressure on the spring. This done I turn the screw, P, until the upper surface of the spring is exactly $\frac{11}{20}$ of an inch from the lever. Having arrived at this point, the permanent adjustment is complete, and the screw must not be touched. I then place 300 grammes instead of 100 in the pan, and ascertain the distance between the surface of the spring and the lever under this increased pressure; and finally, by repeating the experiment with various weights, I

determine the relation between distance and pressure with such accuracy, that by measuring the former the latter can always be known.

The only other modification I have to recommend is, the substitution of smoked glass for paper. The alteration required for this purpose is very trifling. All that is necessary is, to remove from the brass frame on which the slip of paper is carried, the spring by which it is held in its place; and to substitute a needle point for the pen. The glass plate must be held in its place by a clamp, for which purpose a pair of common bulldog forceps answers exceedingly well. The glass plates I employ measure 5 in. by $1\frac{1}{4}$ in. Each plate is smoked on one side by holding it horizontally over the middle of the flame of a spirit lamp trimmed with paraffin, taking care to move the plate backwards and forwards in a direction at right angles to its length. In this way an even layer is deposited, which should be so thin that a flame seen through should appear of a dull red colour. The advantages of this improvement are manifold, the first being that friction is much diminished, especially if great care be taken in the adjustment of the pen to the proper distance, which can be effected with great accuracy by means of the steel screws on which the lever works. A second advantage is the avoidance of failure in the record. By the old method this

failure was not infrequent; it often happened that the pen would not work, either because it was out of order, or because the paper was greasy, or the ink not sufficiently fluid. The needle point, on the contrary, never fails to leave a clear and sharp result. The tracings may be preserved by varnishing them, for which purpose the method ordinarily employed by photographers is best adapted. These permanent records have two advantages over those on paper. In the first place, they may be readily exhibited by means of the magic lantern without any further preparation; and secondly, they can be readily reproduced by photography.

Mode of applying the sphygmograph to the wrist.—Great care should be taken that the spring is always applied in the same situation, and that that point is selected at which the artery is most favourably placed for the exploration of its expansive movements. The part of the radial artery which is ordinarily felt by the finger may be divided into an upper and a lower portion, the line of demarcation between them being the transverse ligament which extends across from the most projecting part of the styloid process to the ulna. The artery can be best explored just as it passes over the ligament, which can generally be felt by the finger; for above it is surrounded by a quantity of fatty cellular tissue, and lies on the surface of the *pronator*

quadratus muscle, whereas beyond, in the interval between the ligament and the scaphoid prominence (*eminentia carpi radialis*), it sinks below the tendon of the *flexor longus pollicis*. In order that the centre of the convex ivory plate which shields the end of the spring may press on the artery at the point indicated, the best rule to follow is to make the edge of the block next the spring coincident with a line drawn across the wrist from the radial spine, while its inner edge rests upon the tendon of the *flexor longus pollicis*, and on the prominence of the scaphoid.

Mode of taking an observation.—The instrument having been properly adjusted, two tracings should be taken, one with a block of $5\frac{1}{2}$ lines vertical measurement, the other with a block of $3\frac{1}{2}$. The comparison of the two results will enable you to form a conclusion as to the resistance offered by the artery to compression, when, if it appear desirable to pursue the investigation, the pressure may be still further increased by reducing the vertical measurement of the block to $\frac{1}{8}$ of an inch = $1\frac{1}{2}$ line. I find by experiment with my own sphygmograph, that by using blocks of the thicknesses indicated, I can produce variations of pressure amounting to 200 grammes, for the determination of which, however, I rely not on the depth of the blocks, but on the measurement of the interval between the spring and the wooden lever.

CHAPTER II.

THE PULSE.

Terms usually employed in describing the character of the pulse as felt at the wrist.—The following characters are described by authors as distinguishable by the finger.

1. As regards the number of pulsations per minute, the pulse is said to be frequent or infrequent,—*pulsus frequens, pulsus rarus.*

2. As regards the time which seems to be occupied by each beat, not including the interval between it and its successors, the pulse is said to be slow or quick (see Dr. Guy, in 'Physician's Vade Mecum,' p. 115)—*pulsus celer, pulsus tardus* (Ludwig, Vierordt, and other writers).

3. As regards the degree in which the artery dilates in length and breadth, the pulse is said to be large or small—*pulsus magnus, pulsus parvus.*

4. As regards the compressibility of the artery, the pulse is said to be hard or soft—*pulsus durus, pulsus mollis.*

It is not necessary to dwell upon the first of these

characters, for the number of pulsations per minute can be estimated with quite as much accuracy by the finger as by the sphygmograph. There are, however, certain rare cases, in which its testimony on this point may be of great value, particularly those in which the frequency of the pulse varies within short periods of time, or is affected by the respiratory movements of the chest.

2. *Pulsus celer, pulsus tardus.*—These two words, or their equivalents, have been used from a very early period in the history of medicine to designate the duration of the arterial expansion. Thus the English word 'quick' was applied by the writers of the last century, not in the loose sense in which we employ it, as if it meant the same as 'frequent,' but to denote that condition of the circulation in which the heart accomplishes its contraction almost instantaneously. There are more reasons than one why the expression has fallen into disuse. The first and best is, that it is very doubtful whether the quality of celerity can ever be truly appreciated by the finger. The old pathologists rightly reasoned, that inasmuch as it can hardly be supposed that the duration of the arterial pulsation is the same in all individuals, such qualities as celerity and tardiness must exist, and therefore set themselves to discriminate them by the finger. It is not wonderful that they succeeded; for, in an age in which the *tactus eruditus* was more culti-

vated and much more believed in than at present, the physician often thought he felt peculiarities which the most practised fingers of the present day are incapable of distinguishing. However this may be, it is certain that the *pulsus celer* and *pulsus tardus* have shared the lot of the temperaments, the *constitutio morborum stationaria*, and many other true notions of our fathers, which we now reject simply because we have no means of defining their significance. The doctrine of temperaments will, I confidently believe, be revived as soon as our newly-born physiological pathology has developed to something like maturity; for it has been lost sight of, not because the ancients were wrong in imagining that different individuals had different temperaments, but because we are so in the habit of insisting on exactness of definition that we cannot content ourselves with the old generalities. So also as regards the *pulsus celer* and *pulsus tardus*; they have been set aside as over refinements by the most judicious writers of modern times,* simply because it was not certain what was meant by the terms by those writers who used them. The sphygmograph, however, enables us not only to understand, but to measure the qualities they express, so that now we are justified in reviving their use. In doing so

* See, on this point, Sir Thos. Watson's Lectures, vol. i. p. 130.

it is probably not desirable again to employ the words 'quick' and 'slow,' which are so apt to be confused with 'frequent' and 'rare.' The best adjectives seem to be *short* and *long*, the familiar use of which in prosody in a perfectly analogous sense, seems to me a good reason for adopting them in sphygmography.

The diseases of which the *pulsus celer* was supposed to be most characteristic, were phthisis and hysteria, in both of which the pulse was said to be *frequens, celer et parvus.* In inflammatory fever the pulse was described as *frequens, magnus et celer*, differing from that of plethora, which was *magnus et tardus.*

3. *Pulsus magnus, pulsus parvus.*—The characters of largeness and smallness are even more difficult to appreciate and more apt to be mistaken without instrumental aid, than those of duration. Nothing, for example, is more common than for a pulse to be described as large, merely because the artery itself is dilated, as is so often the case in advanced life. So also in the bounding, short pulse, a mistaken impression of largeness is apt to be conveyed to the finger.

The *pulsus magnus* is understood to mean that state of the circulation in which a full wave of blood passes along the artery at each stroke, the artery dilating largely in length and breadth, acquiring a greater calibre than before, and becoming at the

same time more or less twisted. This form of pulse has been supposed to characterise the eruptive fevers; it is the *pulsus frequens, magnus et mollis*, from which that of inflammatory fever differs in possessing the quality of hardness. In apoplexy and compression, the pulse was said to have the same quality as in inflammation, with the exception that it was less frequent. The small pulse occurs, as has already been said, in phthisis, and in all exhaustive diseases towards the close of life.

4. *Pulsus durus, pulsus mollis.*—Hardness is the character that used to be considered as the warrant for bleeding, the pulse of inflammatory fever being described as *frequens, magnus, durus et celer.* What this incompressible, bounding pulse was, we have no opportunity of investigating experimentally; for nowadays it is never met with. Sir Thomas Watson, writing in 1848, tells us that 'it does not occur in all inflammations, and may occur where there is no inflammation. It may depend upon hypertrophy of the left ventricle of the heart,' or, on the other hand, 'may be connected with a morbid condition of the artery itself, brought on, as Dr. Latham has suggested, by the pernicious habit of dram drinking.'* From this passage I infer that the inflammatory pulse conveyed the same feeling to the finger as the well-known pulse of hypertrophy of the left ventricle,

* Lectures, vol. i. p. 131.

to the sphygmographic characters of which I have directed attention in my lecture. The observations I have made as to the impossibility of accurately measuring by the finger the qualities of duration and amplitude, do not at all apply to those of resistance. As Sir Thomas Watson observes, 'the finger requires a certain education for the purpose,'* but the faculty is one which may be acquired by most persons who are willing to take the requisite pains.

Of soft pulses several varieties have been described by authors. Thus the small and soft pulse is met with towards the close of slowly progressing exhaustive diseases. The large and soft pulse occurs in chlorosis, especially those cases which are characterised by great transparency of skin. But the most common example is that of mild febrile reaction. This is met with at the outset of many acute diseases and particularly of the eruptive fevers, during the stage which immediately follows the rigor. So long as the rigor lasts, the pulse has the character which is described by the terms wiry or thready—*pulsus frequens, parvus, celer et durus.* When reaction sets in, it changes from hard to soft, and undergoes an alteration of frequency, with reference to which experience teaches that, in proportion as the pulse becomes slower as well as softer and more

* Lectures, vol. i. p. 219.

expansive, the patient feels relieved and his condition is improved.

Arterial movements which constitute the pulse.— In the preceding section we have seen that great uncertainty exists as to the precise meaning of many of the terms which have been used by medical authors in describing the tactile varieties of the pulse. This vagueness of definition was unavoidable so long as no means existed of measuring the duration and intensity of the alternate movements of arterial expansion and relaxation, and comparing the results in different cases. The facts relating to these movements are set forth in my lecture. As a further aid to the clear comprehension of them, I now present them to the reader in a summary form, requesting him to bear in mind, first of all, that just as the appearances of the tongue are of use only in so far as they reveal the condition of the digestive mucous membrane generally, so the pulse must be constantly studied in relation to the contracting heart which produces it. The days of pulse divination are long past. In certain rare instances we may find out what is the matter by feeling a patient's pulse, just as by looking at his countenance, but in general we may be content if by means of it we can discover the condition of his circulation. The following facts are, I believe, made out with tolerable certainty and distinctness.

1. At the moment that the heart begins to contract,

a vibratory movement of the blood contained in the aorta is produced, in which the molecules of liquid are projected forwards in the axis of the vessel. A similar vibratory movement occurs at the moment that the ventricle ceases to contract. These two movements differ only in this respect, that in the former the primary shock is directed towards the periphery, in the latter towards the heart.

2. Each of these movements is propagated in the direction of the stream at a rate of about ninety feet per minute—the one expressing itself at the radial artery by a sudden expansion of the arterial tube, the other by a sudden collapse.

3. The exact moment at which each movement occurs is indicated by the sphygmograph—the former by a sudden vertical ascent of the lever, the latter by a descent. In either case the primary movement is followed by a succession of smaller movements in alternately opposite directions. By measuring the time which intervenes between the first and second vibration, the exact duration of the systole of the heart may be ascertained; for the one occurs at the instant that the ventricle hardens in contraction, the other at the instant that the aortic valve becomes tense in closing.

4. Both movements manifest themselves with much greater distinctness in some cases than in others. In this difference it seems probable that the respective

valves concerned have much to do, and that the systolic vibration is produced by the sudden tightening of the mitral valve, just as the diastolic vibration is due to the sudden tightening of the aortic valve. The precise physical conditions on which the degree of vibration depends, have not as yet been ascertained, but observation points to the conclusion that the intensity of the systolic vibration is greatest when the arterial pressure is lowest at the close of the diastolic period.

5. From the moment that the ventricles begin to contract, the fulness of the arterial system and, consequently, the arterial pressure, rapidly increase. As, however, the arteries at first yield readily to the tide of blood, the tension does not attain its maximum until some time after the hardening of the ventricle in contraction. The duration of the interval between the one event and the other—that is, between the closure of the mitral valve and the moment of highest pressure in the radial artery—varies. It is longest when the arterial system is full; shortest when it is comparatively empty. Hence the measurement of this interval comes to be of considerable importance.

6. From the moment that the artery attains its greatest distension it begins to collapse; the form of that part of the sphygmographic tracing which corresponds to the period of relaxation, is, as has

been recently pointed out by Dr. Divers, parabolic.* In the normal pulse the parabolic form is not easily distinguished, the line of descent being nearly rectilinear; but in all those pulses in which the collapse is rapid, it is very obvious, and most of all in what has been called the monocrotous form, which corresponds to the thready pulse of authors.†

7. In certain conditions of the circulation the radial artery, immediately after the distension produced by the contraction of the left ventricle, suddenly collapses, and then as suddenly expands again —the second expansion being sometimes nearly equal in intensity to the first. This constitutes dicrotism. Considering its importance as a sign of disease, and that its pathological relations cannot be understood without analysing its phenomena, I shall endeavour, even at the risk of repetition, to sum up the more general description which I have given of them in my lecture.

The phenomena in question take place during the diastolic relaxation of the heart. As, however, they are affected by what occurs in the arteries immediately before the aortic valve closes, it is necessary to commence the description from the moment that the ventricle contracts. This contrac-

* British Medical Journal, Aug. 3, 1867.

† See p. 26.

tion produces expansion of the arteries, and acceleration of the progressive movement of their contents. These two associated and simultaneous effects are not only much more marked in the large arteries than in the small, but occur at an earlier moment, so that at the periphery the current attains its greatest acceleration *somewhat later* than near the heart. Hence, at the moment that the ventricle relaxes and the influx of blood through the aortic valve ceases, blood is still moving rapidly onward in the small arteries. As a necessary result, the arterial system becomes relaxed, and the progressive movement of the blood is retarded, collapse beginning where the stoppage occurred (viz. at the aortic valve), and being propagated towards the periphery. Then, as the capillary arteries are relaxed, the capillary circulation is retarded, while the aorta becomes simultaneously distended in consequence of the increased resistance in front. This distension is in its turn propagated towards the periphery, and is succeeded, like the systolic distension, by collapse. If the conditions are favourable, the same series of movements may be several times repeated, the differences between the alternating conditions becoming less and less at each repetition. Thus we have the following succession of phenomena :—

1. Contraction of the left ventricle.

2. Distension of the aorta, and greatest acceleration of blood stream in the great arteries.

3. Greatest acceleration in the peripheral arteries, occurring simultaneously with cessation of progressive movement in the aorta.

4. Diminished distension, and diminished progressive movement in the aorta.

5. Propagation of these effects to the capillary circulation, and consequent increase of arterial resistance.

6. Propagation of the resulting aortic distension towards the periphery, producing peripheral acceleration, and so on.

Admitting the above to be a correct account of the phenomena, it is obvious that dicrotism must stand in the closest relation to the velocity of the blood stream in the peripheral arteries at the moment that the heart ceases to contract. This is true, both as regards the collapse which intervenes between the first and second beat, and the second beat itself—the diastolic expansion or fourth event. For the diastolic collapse depends entirely on the fact that at the moment that the influx of blood from behind is suddenly cut off, blood is rapidly flowing through the capillaries to the veins, so that the more rapid the efflux the more complete the collapse. In other words, the intensity of the diastolic collapse does not, as often stated, depend on diminished pressure directly, but on the degree in which the systolic acceleration is

propagated towards the periphery. Its apparent relation to tension may be easily understood when it is considered, that it is only when the arterial system is empty that this propagation can take place. In general the rate of movement near the periphery is nearly the same during systole, as during diastole; but in proportion as the pressure in the small arteries approaches that of the venous system, the effect of the heart's contraction on the former becomes more obvious; for the *difference* between the systolic and diastolic arterial pressures, even though it may be absolutely no greater when the arterial system is empty than when it is tense, is much greater in proportion to the mean pressures existing in the two systems at the same time. When the arterial pressure indeed is very low, the circulation may be even arrested during the diastolic interval, owing to the arterial tension scarcely exceeding the venous.

The same considerations serve in explanation of the conditions on which the second beat depends. The diastolic expansion occurs simultaneously with that arrest of the progressive movement of the blood in the peripheral arteries, which is the immediate result of the diastolic collapse. It is obvious that the same condition (viz. the approximation of the arterial to the venous pressure during the repose of the heart), which determines the one effect, is also favourable to the other. Thus in the undulatory

double pulse of typhus, the intensity and suddenness of the second expansion appears nearly as great as the first, the explanation being, that in this condition the contractions of the heart are extremely feeble; the arterial pressure being so low that progressive movement of blood ceases entirely in the capillaries during the diastolic interval.

From the preceding considerations it follows that dicrotism is characteristic of that condition of the circulation in which the arterial pressure is diminished, while the venous is increased. It denotes that the capillary current, instead of being constant in its rate of movement, is markedly accelerated during diastole, and retarded during the diastolic interval.

This seems to be the place for pointing out the distinction between the dicrotous pulse and the so-called wiry or thready pulse of authors—*pulsus durus et parvus*. When the pulse is small and hard, as during the rigor at the onset of acute diseases, and in certain dangerous forms of carditis, the expansion of the radial artery is sudden, and of short duration; the suddenness of the movement not depending upon the rapidity with which the arteries empty themselves by the capillaries, but on the violence with which the heart itself contracts. In this form of pulse there is no second beat. The explanation is clear; the difference between the

arterial and venous pressures is so considerable, and the range of variation in the peripheral arteries so limited, that no perceptible diastolic retardation takes place in the capillaries, and consequently no second expansion.*

Practical application of results.—The sphygmograph is not to be regarded, like the laryngoscope or the ophthalmoscope, as an aid in the discovery and discrimination of organic diseases, for affections the most diverse communicate to the pulse the same graphical characters. Its use is to enable the physician to investigate the state of the circulation and circulatory organs in diseases of which the general nature is already recognised, with reference to (1) the mode and duration of the contraction of the heart; (2) the soundness of the arteries; and (3) the relative quantity of blood contained in the arteries and veins, or, in other words, the balance of pressure between the venous and arterial systems. If it can be shown that the sphygmograph affords a reliable means of determining these most important conditions of disease, it is quite enough, without forcing it to applications of which it is from its very nature incapable.

1. The heart possesses in itself the power of con-

* The condition of circulation indicated by the thready pulse is analogous to that produced in animals by section of the pneumogastric nerves.

tracting rhythmically. This faculty may be exercised either automatically, i.e. independently of the central nervous system; or partly automatically, partly under the influence of impressions reaching it through the spinal cord. The researches of Bezold have shown that the nerves through which these impressions are conveyed are those which, originating from the spinal nerves and passing through the thoracic and lumbar ganglia of the sympathetic, converge to the posterior cardiac plexus. The same physiologist has also shown that it is through these channels that the modifications of the mode of contraction of the heart which result from emotion and suffering are induced, and render it probable that the perverted actions of the heart met with in fever and other acute constitutional disorders are also spinal in their origin.

A number of facts seem to make it probable that, *whenever the heart contracts of itself*, i.e. *automatically, it contracts gradually and peristaltically, its constituent fibres being brought successively into action; and that, in so far as the movement is deprived of its automatic character by the influence of stimuli acting through the spinal cord, it becomes sudden and instantaneous.*

Sphygmographically, suddenness of contraction manifests itself in verticality and amplitude of the primary ascent of the tracing; while in those forms

of pulse which correspond to a more gradual mode of contraction, the first event is indistinguishable. Hence we are led to associate absence or suppression of the first event with all those conditions of the circulation in which the heart may be supposed to act automatically; and to believe that, whenever the artery expands sharply under the finger at the moment of the shock of the heart—whenever, in short, the first event becomes a prominent feature of the tracing — we have evidence therein that the systole of the ventricle is no longer peristaltic, but reflex; and that, through the spinal cord, influences are at work (whether originating from emotions, sensations, or abnormal constituents in the blood) which are not altogether normal. In all such cases, a sharp sound, having the character of the second sound, is heard on auscultation in the neighbourhood of the præcordial impulse.*

2. *Absence of elasticity* is inseparably associated with hypertrophy and dilatation of the arteries, and increased arterial resistance. In the natural state of the circulation, the increased tension produced in the arteries by the ventricular systole manifests itself partly during the systole itself, partly during the succeeding pause; the agent in thus distributing the effect being obviously the arterial elasticity. When

* To this sound I am accustomed to apply the term 'shock sound.' See p. 41.

the arteries are not elastic, this distribution of tension does not occur; and consequently, we find the arteries relatively more tense during systole, and more relaxed during diastole. Further, the increased resistance prolongs the systolic period. Hence, as may be readily understood, the diastolic collapse is indicated in the tracing by a vertical line of greater length than usual, and occurs at a later period. The length of the vertical descent indicates the amount of the sudden diminution of arterial pressure, which coincides with the cessation of the ventricular systole.

3. *Relative fulness of the arterial system.*—Inasmuch as the quantity of blood contained in the circulatory system is more or less constant, it follows that there must be an inverse relation between the quantity contained in the arteries and veins at the same time, so that when the one increases, the other must proportionately diminish. Now, it is the function of the heart, if one may so express it, to preside over this relation, by increasing the vigour of its contractions whenever the arterial pressure becomes insufficient for carrying on the circulation, and relaxing its efforts when the arteries become too full. So important, indeed, is the maintenance of the balance of tension between arteries and veins, that it involves the whole question of the competency of the heart to perform its mechanical functions.

Undue impletion of the arterial system shows itself

in *postponement of the arterial diastolic collapse*, which means, as regards the heart, that, in consequence of increased arterial resistance, the *left ventricle continues full for an unnaturally long period* —not collapsing, indeed, until its muscular contractility becomes exhausted. In this state of the circulation, the first sound of the heart is prolonged up to the moment when the second sound is audible.

Insufficient impletion of the arterial system (supposing the arteries to be in a healthy state) is necessarily associated with *diminished elasticity*, i.e. diminished elastic resistance, or, to use the converse expression, increased elastic yieldingness. It shows itself sphygmographically by a well-defined character —the *absence of the second event*, or true dicrotism, which means that the diastolic collapse occurs immediately after the primary expansion (the vertical ascent of the tracing ending at once in a nearly vertical descent), and that the contraction of the heart, however violent it may be, produces little effect in increasing the arterial tension.

Analysis of the pulse-tracing.—In order to derive from the sphygmographic record all the information as to the state of the circulation that it is capable of affording, it is necessary to adopt an invariable method, in selecting which it is to be borne in mind, that the two most important characters are those of duration and resistance to

pressure. The elements to be determined in every observation are the following: 1. The relative duration of the systole and the diastolic pause; 2. The duration of the systolic distension, and the interval between it and the diastolic distension; 3. The intensity of the systolic and diastolic vibrations; 4. The effects of varying the tension of the spring of the sphygmograph.

The duration of the systole is readily measured in vibratile pulses by measuring the interval between the systolic and diastolic vibrations. If these are indistinguishable it is to be done by measuring from the commencement of the ascending limb of the tracing to that point in the descending limb at which the line is interrupted. If this cannot be made out, accurate measurement is impossible. For the determination of the duration of the systolic distension, by which I mean the interval of time which elapses between the commencement of expansion and the moment of greatest distension, a pressure of about 300 grammes should be employed. The period can then be correctly estimated, and should be measured from the commencement of the ascending limb to the vertical line which passes through the point of greatest elevation in the tracing. The same method is applicable to the measurement of the interval between the first and second beat. And lastly by observing the effect of variations of pressure we are enabled to

PLATE I. SOFT PULSES.

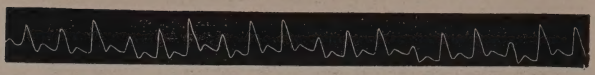


FIG. 1.— Pulse of irritative fever.
(Pressure of spring, about 130 grammes; Frequency, 190 per minute.)

FIG. 2.—Undulatory pulse of typhus.
(Pressure, about 160 grammes; Frequency, 160.)

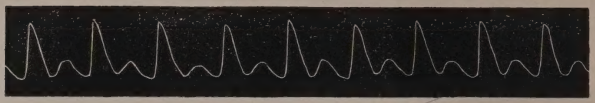


FIG. 3.—Soft and frequent pulse of mild pyrexia.
(Pressure, about 160 grammes; Frequency, 90.)

FIG. 4*a*.—Normal soft pulse.
(Pressure, about 160 grammes; Frequency, 58–60.)

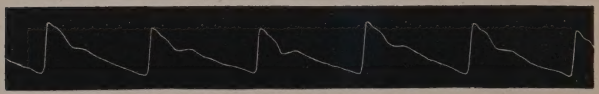


FIG. 4*b*.—Pulse of the same person after exercise and residence in the country.
(Pressure, about 160 grammes; Frequency, 56.)

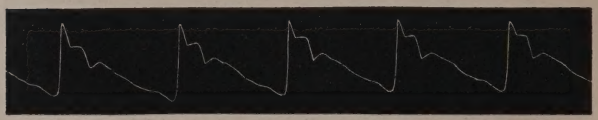


FIG. 4*c*.—The same. (Pressure, 90 grammes; Frequency, as before.)

PLATE II. HARD PULSES.

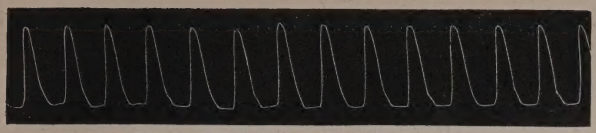


FIG. 1.—Wiry pulse of rheumatic carditis.
(Frequency, 150; Pressure of spring, 170 grammes.)

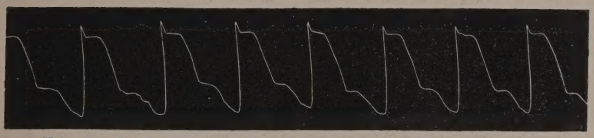


FIG. 2.—Hard and long pulse of hypertrophy of the left ventricle, with dilatation. (P. durus, magnus, et tardus.)
(Frequency, 84; Pressure, about 170 grammes.)

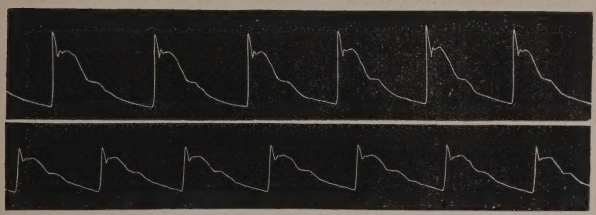


FIG. 3.—Hard pulse of chronic Bright's disease (contracted kidney).
(Frequency, 70; Pressure of spring—upper line 300 grammes, lower line 150 grammes.)

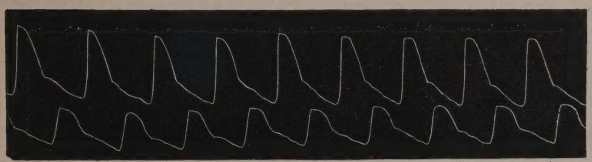


FIG. 4.—Hard pulse of Bright's disease, similar to that represented in FIG. 3 but less vibratile. (Frequency, 96–100; Pressure—upper line 300 grammes, lower line 90 grammes.)

FIG. 5.—The firm and long pulse of vigorous health.
(Frequency, 50; Pressure, about 170 grammes.)

[*To be inserted after p.* 33.

distinguish between hard pulses and soft; and in doubtful cases to discriminate between those features of the tracing which express vibrations and those which indicate arterial distension, the former being, for the most part, suppressed by strong pressure, whereas the others are unaffected or rendered more prominent.

Classification of pulses.—The forms of pulse met with in health and disease may be divided into two great groups—the soft or atonic, and the hard. These may be further subdivided according to duration and expansive movement. For practical purposes the following classification will be found sufficiently minute. (See Plates I. and II.)

Soft Pulses.

1. The pulse of irritative fever. (Pulsus mollis et celer.)
2. The undulatory * dicrotous pulse. (Pulsus mollis, parvus et tardus.)
3. The large and soft dicrotous pulse. (Pulsus mollis et magnus.)

Hard Pulses.

4. The small collapsing or thready pulse. (Pulsus durus, parvus et celer.)
5. The pulse of hypertrophy. (Pulsus durus, magnus et tardus.)
6. The large collapsing pulse or pulse of aortic regurgitation. (Pulsus durus, magnus et celer.)
7. The pulse of vigorous health. (Pulsus durus, parvus et tardus.)

* The word 'undulatory' has been used in a somewhat different sense by Dr. Anstie. See *Lancet*, Sept 1867.

CHAPTER III.

NOTES ON CERTAIN CAUSES AND COMPLICATIONS OF DISORDERS OF THE CIRCULATION.

UNDER THIS HEAD I propose to bring together various conditions of disease, some anatomical, others functional, which have little or nothing in common excepting that they claim the attention of those who desire to study disorders of the circulation from a physiological point of view, either because they occur as complications of these disorders, or produce them.

1. *Mitral valvular disease.*—The mechanical effect of disease of the mitral valve on the circulation is difficult to judge of by observation; for this affection is so often associated with some other functional disturbance that opportunities seldom offer themselves of studying uncomplicated cases. Experience shows that, with the same amount of mitral regurgitation, the pulse may exhibit great differences of character.

The pulse of mitral regurgitation is rarely of normal *frequency*; the increase is mainly owing to the fact that the left auricle is always full, and is thereby stimulated to frequent contraction by the pressure of blood on its internal surface.

The *duration* of the systole of the heart is always diminished when the regurgitation is considerable. It is, however, difficult to measure, from the absence of vibratile characters. The systolic expansion is always short, and the systolic arterial tension low. So long as the pulse is regular, its brevity and compressibility afford the most constant indications of the amount of regurgitation that exists.

The *irregularity* met with in mitral disease possesses a character not before noticed by writers, which affords a key both to its cause and the only mode of remedying it. In the irregular pulse of mitral regurgitation the contractions of the heart may be divided into two classes—those which are frequent, short and ineffectual, and those which are long and forcible; and it is found by observation, that the former occur during inspiration, the latter during the respiratory pause. It is further found that certain dyscrasiæ produce a mode of irregularity, identical with that of mitral disease in every respect, and particularly in its relation to the thoracic movements. Thus we have the same results produced in

the one case by a dyscrasia and in the other by a mechanical defect, and the question arises, What is the relation of the one to the other?

The mechanical effect of inspiration, as I have elsewhere shown,* is to augment the quantity of blood contained in the pulmonary circulation, and hence to increase the frequency of the contractions of the heart. This increased frequency depends on the distended state of the auricles, in consequence of which the ventricles fill more rapidly during their period of relaxation. In this way the length of the diastolic pause is diminished and the hurried action of the heart satisfactorily accounted for; but the question still arises, Why are the rapid beats which occur in inspiration also ineffectual? Very probably because the mitral valve does not close; the heart being distended with blood its walls are kept apart to such an extent that the curtains do not meet. The ventricle contracts, but much of its blood is discharged into the auricle, to be returned to the ventricle as soon as its contraction is over. It is not until the effect of inspiration in keeping the auricles full ceases, that the curtains get near enough to allow the heart to make an effort sufficiently effectual to send a full tide of blood into the aorta, and thus relieve the distended pulmonary circulation.

* Croonian Lecture for 1867. Proceedings of the Royal Society, and Phil. Trans.

This theory, more detailed proofs of which I hope to be able to furnish at a future period, serves to explain all the phenomena of mitral irregularity which have come under my notice. It is in complete accordance with the fact that in those forms of disease in which the incompetency arises not from defect of the valve itself, but from permanent enlargement of the orifice, the pulse has constantly the frequent, short, and ineffectual character, which in less degrees of regurgitation it possesses only during inspiration.

The effect of mitral *stenosis* is to diminish the quantity of blood discharged by the heart at each contraction, by preventing the repletion of the left ventricle during the diastolic pause. So long as the patient is kept absolutely tranquil, and the processes of life can be carried on with a scanty supply of blood, the heart does its work without difficulty. But, whenever more blood is used in muscular contraction, or when the heart is excited by emotion or any other cause of functional disturbance, the danger becomes imminent, death taking place in fatal cases by sudden syncope, consequent no doubt on anæmia of the nervous centres. In this most dangerous form of heart disease, the comparison of the state of the pulse before and after exertion, would probably be of value in prognosis.

2. *Aortic valvular disease.*—In the slighter degrees of aortic regurgitation the sphygmographic

characters of the pulse are only in so far altered as the left ventricle is hypertrophied; for which reason the exploration of the pulse is not to be compared for a moment to auscultation as a means of diagnosis. Copious regurgitation, however, is indicated by very obvious alterations. The contrast between its effect and that of mitral incompetence consists in this, that while the one occurs during systole, the other occurs during the diastolic pause. Both tend to render the work done by the heart unproductive, and consequently to diminish the arterial pressure; but in the one case the leakage is from the distended aorta into the relaxed ventricle, in the other from the distended ventricle into the still contracting auricle. On the whole, aortic regurgitation, regarded merely as a mechanical defect, is more serious than mitral regurgitation.

The peculiarity of the aortic pulse consists in the complete emptiness of the artery during the diastolic period, and the extreme rapidity with which the systolic distension is propagated to the wrist; and here it is to be noticed, that the vertical rise of the lever has in this case a somewhat different meaning from that which attaches to it when observed in the normal condition of the circulatory apparatus. For in the aortic pulse the systolic vibration can hardly be distinguished from the distension. In other words, the arterial pressure attains its maximum in-

stantaneously; so that the sudden expansion of the artery is not to be regarded as a merely vibratile phenomenon. That this is the case we learn, by observing that the verticality of the ascending limb of the tracing is not diminished by increasing the tension of the spring of the sphygmograph.

The characters communicated to the pulse in aortic stenosis are similar to those observed in hypertrophy of the left ventricle, the distinctive peculiarity being, that the time occupied in the contraction of the heart is lengthened, and that the expansion of the arteries takes place slowly and gradually.

3. *Rheumatic carditis.*—The pulse which indicates that the heart is becoming affected in acute rheumatism, is shown by the sphygmograph to be one simply of excitement. The ventricles contract with violence, but the duration of contraction is short; the arterial tension is excessive, and the pulse frequent, short, hard, and vibrating. This kind of pulse is regarded by many physicians as a sign of pericarditis; it is open to question whether the disorder which it indicates may not rather have its seat in the muscular fibre.

4. *Evidence of increased arterial resistance, to be derived from the exploration of the præcordial impulse, and by auscultation.*—I have elsewhere fully described the characters communicated to the radial pulse when the energy of the heart is insufficient to

overcome the arterial resistance. I have now to advert to the further indications of the same state of the circulation which are to be obtained by the exploration of the movements of the heart itself. With the *pulsus tardus* a laboured mode of contraction of the heart is associated, which, although it must have been unconsciously recognised by physicians, has not been described. The præcordial impulse of the overdone heart is not only expansive but unduly prolonged. This signifies that the globular form, which is normally assumed at the moment that the ventricles harden, is retained throughout the systolic period, the efflux being so slow and difficult that the organ does not empty itself thoroughly as it ought to do, retaining a quantity of blood in its cavity, even at the end of its contraction.

The next character is of equal importance, and even more easily recognised. The end of the systole, which in the healthy heart is not felt at all, is marked in the exhausted heart by an obvious shrinking, which is often sensible not only at the impulse, but over the whole præcordial region. In listening to such a chest it is observed that the first sound, if not accompanied by an abnormal murmur, is unduly prolonged. Very frequently it is what is called reduplicated. This signifies, not that the two ventricles contract at different moments, but that two kinds of sound are produced. At the instant that the ventricle

hardens, a sharp sound is heard, which I am in the habit of calling the shock-sound. It is without doubt synchronous with the closure of the mitral valve, and is, I believe, produced by it. The other is a dull sound, and lasts during the whole systole, ending suddenly in the second sound.

Thus, as regards the heart, the signs of undue resistance are: 1. Prolonged præcordial expansion, commencing with the impulse, and terminating in sudden collapse; and, 2. Prolonged first sound, often commencing with the sharp shock-sound, and ending in an accentuated second sound.

In order to interpret rightly these signs, it must be borne in mind that the strain on the heart may arise from various causes. In the absence of valvular obstruction, the seat of disorder may be either the arteries or the heart itself. In the former case it may be referable either to the capillaries, as in chronic albuminuria, or to the larger trunks, as in senile degeneration, when the energy of the heart is wasted in consequence of the loss of arterial elasticity. In the latter case it may arise from interstitial change in the muscular fibres of the heart itself, rendering it more or less incapable of accomplishing its functions, and tending to the production of dilatation.

In the gradual progress of the disorder of the circulation which is induced by chronic Bright's

disease, two stages may be always distinguished. During the first period, commencing from the time that the patient begins to experience præcordial distress or palpitation, the heart contracts with undue energy, the pulse being always hard and resistant. The second stage, on the other hand, is characterised by dilatation of the left ventricle, arising principally from the extreme pressure to which its internal surface is exposed in contracting, occasionally perhaps from degeneration of its muscular tissue. As the disease progresses, the heart becomes more and more incapable of performing its functions, its gradual loss of energy being indicated by changes in the pulse-tracing from that which I have specially described in my lecture as characteristic of hypertrophy, to that of atony.

5. *Respiratory and pulsatile movements of the veins of the neck.*—Just as the radial artery affords, from its peculiar anatomical relations, the best opportunity of ascertaining the state of the arteries in general, so the state of the venous system, and of the right auricle of the heart which forms a part of it, can be best judged of by the exploration of the jugular vein and its tributaries.

Two kinds of movements are observed in disease in the veins of the front of the neck, which, although often confused with each other, differ both in their nature and mode of production. One of them is synchronical with the thoracic movements, and may

therefore be designated respiratory; the other is produced by the contractions of the heart, and constitutes the venous pulse. In many cases of cardiac dyspnœa both co-exist.

The respiratory movement can scarcely be understood without referring to the anatomical relations of the parts. The internal jugular vein of the right side lies behind the triangular interval between the sternal and clavicular portion of the sterno-cleido-mastoid muscle, that of the left being somewhat further out. Up to the point at which the vein is crossed by the omo-hyoid muscle, it is much wider than higher up; it is this wide part, named *bulbus venæ jugularis*, in which the movements in question take place. The subclavian veins are enlarged in a similar manner near their terminations in the *venæ anonymæ*, neither jugulars nor subclavians containing any valves. At its commencement, the *vena anonyma* of each side, which lies behind the sternal end of the first rib, is fixed in its place by the deep layer of the *fascia colli*. From the firmness with which its external surface is attached to this structure it is constantly kept open, and at this point it is furnished with a couple of valves, by which the regurgitation of blood into its tributaries is prevented. So long as the vein remains in its natural state these valves perform their functions completely, but it has been shown, by the researches of Hammernik, that

even in healthy persons, as life advances, the jugular and subclavian bulbs gradually dilate to such an extent as to render the curtains of the valves too short to close the aperture. In persons subject to protracted dyspnœa this dilatation proceeds much more rapidly, in consequence of the increased venous pressure produced during forced expiration. In such individuals the cavities of the jugulars are in constant communication with the right auricle, which, from being contained in the thorax, is subject to variations of external pressure due to the respiratory movements. So long as the breathing is tranquil, these do not interfere with the venous circulation, notwithstanding the incompetency of the valves; for the intra-thoracic pressure is always considerably less than that of the atmosphere. But in dyspnœa, even of moderate intensity, the relation between the atmospheric and the intra-thoracic pressure is modified. In inspiration, the pressure to which the right auricle is exposed is much diminished, while in expiration it is correspondingly augmented—so much so, that it not only exceeds that of the atmosphere, but even that of the systemic veins. Hence it arises, that those veins which are in free communication with the auricle are distended during expiration, and collapse during inspiration.

The venous pulse is produced either by the contractions of the right auricle, in which case it is so

slight as to be scarcely perceptible, or by the contractions of the right ventricle, when it is much more strongly marked and obvious. In the former case it occurs about a tenth of a second before the shock of the heart, whereas in the latter it is synchronical with the shock, and is preceded by a fainter beat due to the contraction of the auricle. The feeble auricular beat *before* the shock of the heart signifies nothing more than venous dilatation and incompetency of the venous valves, but the more strongly marked ventricular beat indicates tricuspid regurgitation, and renders it probable that the right side of the heart is dilated.

6. *Influence of consciousness on the circulation.*— The consciousness of painful or even disagreeable impressions has a marked effect on the circulation. Thus, long-continued pain, such as that of toothache, excites the action of the heart, giving rise, in many persons, to a condition of so-called feverishness, although the other elements of fever are absent. An effect of the same kind is associated with that general disturbance of sensibility which is often experienced at the onset of acute diseases, and which is denoted by the French word *malaise*. In either case, it may be sometimes difficult to distinguish between the effects of a painful or disagreeable impression, and those of other diseased conditions which accompany it; but experience and observation seem to me to

teach plainly that the circulation may be excited by pain without the co-operation of any other cause. In the opposite condition to *malaise*, that in which no uncomfortable feeling whatever is experienced—in that quiescent state in which the luxury of doing nothing seems to constitute the height of enjoyment—the heart contracts automatically. Its movements are tranquil and infrequent, and the circulation is in much the same condition as in sleep.

Emotion affects the pulse even more immediately than sensation. Unfortunately, our knowledge of the subject is very indefinite. It is usual to regard some emotions as exciting, others as depressing. The exciting emotions are those which are called impulses, from their impelling the individual to action. Of these, anger, revenge, love, hatred, and even fear in some of its aspects, are instances, all of which, in so far as they are impulsive, excite the heart to more frequent and vigorous contraction. The only purely sedative passion is sorrow—and sorrow only in so far as it is unmixed with any impulse, whether of anger or even despair. It is only that kind of sorrow which is absolutely aimless, that retards and relaxes the contractions of the heart, at the same time that it diminishes the expansive movements of the chest, and lengthens the respiratory pause. Yet it is not incorrect to speak of any of the active emotions as depressing, when long

continued. For any desire or longing, when it possesses the mind for a lengthened period, although at first it excites the contractions of the heart, ends by exhausting and weakening it; and in this way the loss of health which is induced by anxiety, or by that never-ending striving after fruitless activity, with which many persons are tormented, may be in some measure dependent on its effect on the circulation.

LECTURE

ON THE

MODE AND DURATION OF THE CONTRACTION OF THE HEART

IN THEIR RELATION TO THE CHARACTERS OF THE ARTERIAL PULSE IN HEALTH AND DISEASE,

DELIVERED AT THE ROYAL COLLEGE OF PHYSICIANS, LONDON,

MARCH 29, 1867.

LECTURE.

THE ORDER OF SUCCESSION of the muscular movements which take place in the heart each time that it contracts, has been very variously represented by different physiologists. More than a century ago, it was the subject of one of the most celebrated controversies * recorded in the history of medicine. Even now that we profess to be guided rather by observation than by rhetoric, opinions are scarcely less divided. If one notion can be said to be more prevalent than another on the subject, it is that of Haller, who believed that there is a *complete alternation* between the contractions of the auricles and ventricles, and that the time occupied by each cavity in contraction is equal to that during which it dilates or expands.†

* I refer to the discussion between the rival schools of Paris and Montpellier.—See 'Mém. de l'Acad.,' vol. i. 1743.

† Haller, 'Elementa Physiologiæ,' 1757, vol. i. p. 399.

A century before, a truer doctrine had been taught by our own Harvey, who showed that the contraction of the heart is but *one movement*, in which both auricles and ventricles take part, and that during the interval between each contraction and its successor, the heart is absolutely at rest.* This observation of Harvey, whether outshone by the glory of his greater achievement, or merely smothered in the futile disputations of his successors, was certainly forgotten until the discovery of auscultation again drew attention to the subject. By none was its truth more early or more clearly recognised than by our distinguished Fellow, Dr. C. J. B. Williams, who, in his 'Inquiry Respecting the Motion and Sounds of the Heart,' published in 1833, showed that no other view of the mode of the contraction of the heart is consistent with the facts revealed by auscultation. Its most complete vindication is to be found in the classical essay of Dr. John Reid, in the 'Cyclopædia of Anatomy and Physiology,' who gives the following view of the succession of events in a cardiac revolution:—'When the heart of a living animal is exposed, and the organ is acting in a natural manner, the auricles are observed to become distended with blood, then to contract rapidly and simultaneously, and propel part of it into the ventricles; this is accom-

* Harvey, 'Opera omnia,' Lond., 1776, p. 33.

panied with a corresponding enlargement of the ventricles, which is immediately followed by their simultaneous contraction and the propulsion of their blood along the large arteries; then follows a pause, during which the auricles become gradually distended by the blood flowing along the veins. When the auricles are filled, they again contract, and the same train of phenomena just described occurs in uniform succession.'*

Although the researches of later observers, and particularly those of Chauveau, are in perfect harmony with this account, there is a constant tendency to return to the doctrines of Haller and Laennec, which commend themselves to the mind by their extreme simplicity, and consequently to represent the duration of the contraction of the heart as something like half that of the whole period occupied in what is called a cardiac revolution—as being nearly equal, in fact, to the interval between one contraction and its successor.

There are several facts, not difficult of observation, which render this view untenable, and show that the time occupied by the heart in contraction is very much shorter than is commonly supposed. The most obvious, and certainly not the least satisfactory, proof that this is so, is attainable to any one who is capable

* Cyclopædia of Anatomy and Physiology, vol. ii. p. 602.

of measuring short intervals of time by the ear with approximate accuracy, by listening to the sounds of the healthy heart. The first sound of the heart being synchronical with the commencement of the contraction of the ventricle and the closure of the mitral valve, and the second with the closure of the aortic valve, it is obvious that the interval between these two events expresses the duration of the contraction of the heart. Now, the most unpractised auscultator can readily satisfy himself, while listening to the sounds of a heart contracting sixty times in a minute, that the time between the first sound and the second is not equal to that which separates the second from the first, and that it cannot for a moment be admitted (as stated in some of our leading physiological text-books) that the heart occupies half a second in contracting.*

But the most positive and conclusive proofs of the brevity of the systole of the ventricle are obtained by direct observation of the movements of the living heart in animals. Harvey founded his doctrine on the subject on what he saw and felt when the living heart was exposed and seized in the hand. He observed that the heart, in beginning to contract, becomes round and hardens, and that these changes of form and consistence occur with such rapidity as to

* See Dr. Kirke's 'Physiology,' p. 87.

appear instantaneous. In the recent experiments of Chauveau, already referred to, this evidence is rendered more precise, though not more positive, by the introduction of those appliances of measurement which distinguish modern physiological research. Chauveau measured, by a recording dynamometer, the tube of which terminated in an elastic bag introduced into the left ventricle of the horse, the exact time occupied in its contraction, and found it to be about a fifth of a second. The observation of the pulse by the sphygmograph is another method of the same kind; the function of that instrument being to record changes of the same nature as those registered by the dynamometer; for, in the sphygmographic observation, the movements of the heart are communicated to the recording lever by the artery, just as in Chauveau's experiment they are transmitted by the flexible tube.

It is the purpose of the present lecture to show that the sphygmograph, regarded as a dynamometer, is not merely an aid in physiological research, especially with reference to the question of the duration of the successive acts which constitute a cardiac revolution, but that in it the pathologist is furnished with an instrument whereby he can investigate the mode, and measure the duration of the ventricular systole in disease. With this view, I shall endeavour to explain, as clearly as I can, the meaning of the

record inscribed by the lever, or, in other words, the relation between the *form* of the tracing and the *movements* of the heart of which it is a representation.

In every arterial pulsation four events are to be distinguished.

1. *The sudden primary expansion of the artery.*—At the moment when the ventricles contract, all the arteries suddenly expand. This expansion does not, however, necessarily express an increase in the quantity of blood contained in the artery; for it can be produced by any sharp movement, of however small extent, provided that it is of great rapidity and is directed in the axis of tube. It is due to a sudden jerking forwards of the particles of liquid, and is, therefore, of the nature of a commotion or shock, in which each particle communicates its movements to its neighbour—just as when a number of balls are freely suspended in a straight line in contact with each other, a smart rap given to the terminal ball shows its effect, not in any advance of the whole number, but in the jerking forward of the ball at the opposite end of the series.* This primary effect of the heart on the arteries, although so sudden

* To this illustration it was rightly objected by a Fellow, who did me the honour to be present at the lecture, that in the case of the experiment with the balls, the effect in question is not observed unless they be made of elastic material—e.g. of ivory; if they be of clay, the movement of the terminal ball is of no greater extent than of any of the rest, there being no

as to appear instantaneous, is not in reality instantaneously transmitted. By experiments, which will be elsewhere published, I have ascertained that, in elastic tubes containing water, at a pressure nearly equal to that commonly existing in the arterial system, sharp propulsive movements are propagated at a rate of about ninety feet per second, so that the time which intervenes between the shock of the heart and the consequent sudden expansion of the radial artery, probably does not exceed a fiftieth of a second, a period altogether inappreciable without the aid of instruments.*

Thus, the first effect of the contraction of the heart on any artery consists in the rapid transmission of an oscillatory movement through the liquid contained in it in such a way that, although every part of the artery partakes sooner or later of the expansive movement produced, no two parts expand at the

propagation of movement from ball to ball. Water being an inelastic fluid, its particles are analogous to the clay balls, not to the ivory ones. The answer is, that inelastic liquids, when enclosed in elastic receptacles, have the same properties, as regards the transmission of molecular movements, as if they were themselves elastic.

* The laws of transmission of this movement are those which regulate the transmission of all oscillatory movements in liquids enclosed in elastic receptacles, particularly in elastic tubes. They were first investigated by E. H. Weber, to whose essay ('De Pulsu, Resorptione et Tactu.' Leipsic: 1834) the reader is referred for further information.

same moment. From the nature of the movement it further results that it is *repeated*. Each primary expansion of arterial wall at any given point is immediately followed by an equally sudden contraction, and then by a second expansion and contraction of less extent, and these by a third, and so on; so that the artery is thrown into a state of rapid vibration, which is more or less marked, according to the suddenness of the shock of the heart and the elastic yieldingness of the arterial wall.

2. That part of the tracing which corresponds to the *systolic distension* of the artery, i.e. to the transmission to its contents of the pressure existing in the left ventricle during its contraction, can generally be distinguished from the primary ascent, as indicating an event of later occurrence. Whenever it can be so distinguished, it is denoted by an arched line, the concavity of which is downwards. It is that part of the tracing which is most subject to variation. In many varieties of febrile pulse, it is entirely absent; in the pulse of arterial resistance, usually met with in advanced life, it attains its greatest development.

Its physiological significance is great, for it denotes the degree and duration of distension produced by each contraction of the heart, in the particular artery to which the sphygmograph is applied. Pathologically it is no less worthy of attentive consideration;

for it expresses an important quality recognised by the finger in disease, that of *fulness*.* Let us consider what this familiar term signifies. Fulness is supposed to mean, that at each stroke of the heart a large quantity of blood is discharged; or, in other words, that the contraction of the left ventricle is unusually effectual. But this is not quite true, or, if true, does not express the whole truth; for, in all those conditions of the circulation which, with strict accuracy, may be termed *atonic*, the volume of blood which passes through the aortic valve at each beat is large, yet the pulse has a character very different from that of fulness. Again, the fact that fulness of pulse is so frequently met with in old persons cannot be supposed to imply that the contractions of the heart are more effectual towards the close of life than at an earlier period.

The explanation is, that fulness of pulse does not indicate that the artery in which it is produced receives more blood absolutely, but that it receives more in proportion to its capability of disposing of it. The blood which enters the arterial system is disposed of in two ways. Some of it is immediately transmitted through the capillaries, while the

* See the lucid exposition of the characters of the pulse in disease, by Dr. Guy, at p. 115 of the 'Physician's Vade Mecum.' Dr. Guy regards the terms full and large as equivalent—both signifying that the quantity of blood expelled at each contraction of the heart is excessive.

remainder is at first retained within the arteries and added to the quantity previously occupying their cavities, to be subsequently (i.e. during the diastole of the heart) forwarded to the veins. The proportion which the quantity of blood immediately transmitted bears to the quantity which remains in the arteries, depends on the vigour of the contraction of the heart and the state of the contractile capillaries, both of which conditions are subject to great variation. The more readily the capillaries allow blood to pass, the more readily do the arteries empty themselves, supposing that the activity of the heart is unaltered. If, on the other hand, the force of the ventricular systole be increased, the effect produced on the arteries is the same as if the capillaries were narrowed; for they at once become insufficient for the transmission of the quantity of blood thrown into the arterial system by the heart. In either case, there is *delay in the collapse of the artery*, which manifests itself sphygmographically in what I have called the *second event*. The artery is distended, not only at the moment that it first receives blood from the heart, but *continues distended* for a longer or a shorter period afterwards. This continuance of distension has always the same meaning. It denotes the resistance offered by the capillary network to the circulation during the systole of the heart.

3. *The diastolic collapse, or third event.*—In

studying the arterial movements which occur during the diastolic interval or pause, the commencement of which is followed by the closure of the aortic valve, the same distinction is to be drawn between those effects which are due to changes of blood-tension and those which are vibratile. Just as the contraction of the heart produces a sudden, merging into a more gradual, expansion of the arterial system, so the *cessation of contraction* manifests itself in a shrinking or collapse, which is more or less violent, sudden, or gradual, according to the state of the circulation; or, adopting instrumental language, just as the bursting open of the aortic valve is indicated by a vertical ascending straight line, followed by a curve of which the concavity looks downwards, so the movement which leads to the closure of that valve is expressed by a vertical descending line, ending in a curve of which the concavity is upward.

The counter-shock or recoil produced in an elastic tube through which a stream of water is passing, by suddenly arresting the current, is equal in intensity to the shock which occurs when the stream is first set in motion; and the two movements are of the same kind, the only difference being that, in the latter case, the direction of the primary movement coincides with that of the stream, and shows itself in the sudden expansion of the tube, whereas, in the former, it is against the stream, and produces

contraction. If, therefore, the velocity of the blood-stream in the aorta were the same at the end as at the beginning of the systole, the shock at the end of the systolic period would be of the same intensity as at its commencement. The conditions of the circulation are, however, such that this could not possibly occur. For, inasmuch as the left ventricle gradually empties itself of blood, the force with which it compresses its contents gradually diminishes during the progress of its contraction, while the resistance in the aorta increases. Hence, at the moment that the contraction ceases, the rate at which the blood flows through the sigmoid orifice has already undergone considerable diminution.

The collapse of the arterial system being the immediate result of the cessation of the contraction of the heart, the determination of the time at which it occurs is of the greatest importance; for it affords a means of measuring the duration of the contraction. As we shall see further on, the classification of pulses depends upon it, not merely in a sphygmographical sense, but with reference to ordinary characters, recognised by the finger. In investigating the pulse by the finger, the physician judges of its character by the time the artery remains expanded, just in the same way, though not so accurately, as if he measured its duration by the sphygmograph.*

* See p. 14.

4. *The diastolic expansion, or fourth event.*— The fact that every artery again expands after the collapse at the end of the systole, is one which has occasioned much speculation, especially since its constancy in all conditions of the circulation has been known. In adynamic fever, it has been long recognised as communicating to the pulse a special character, that of dicrotism. Its cause has been much discussed. According to Naumann, it is produced by what he calls a recoil of blood against the aortic valve; and the explanation of it given by Marey is not altogether dissimilar, although differently expressed. That such a recoil exists, we have already seen; but that this is the explanation of the subsequent expansion of the artery, I have myself disproved by numerous experiments, to which this is not the proper place to refer more particularly.

What, then, is the cause of dicrotism? Without entering more minutely than is necessary into the physical aspects of the question,* I may be permitted to observe that, from the nature of the phenomenon itself, it implies that the disturbance of pressure-equilibrium in the arterial system which is necessarily produced by the injection of blood from the heart, is excessive. For, if it were otherwise, the arterial distension would subside gradually and uninterruptedly, and be expressed by an unbroken

* The object of dicrotism is more fully discussed at p. 22.

curve, the form of which would correspond to the velocity of the capillary circulation.

Interesting as it would be to follow out these considerations, it is for the present of more importance to study the practical significance of dicrotism as a sign of disease. And this I hope to do satisfactorily by referring to my own observations as to the circumstances under which the dicrotic pulse is observed.

The states of the circulation which produce true dicrotism are just the opposite of those which favour the development of the second event. The one implies fulness of pulse, the other the contrary. The one expresses the continuance of the arterial expansion, the other its abnormal abbreviation. Both alike depend on the proportion which the quantity of blood projected by the heart at each stroke bears to the capillary resistance. The smaller the quantity thrown into the aorta by the ventricle, the sooner is it got rid of by the capillaries, and the more dicrotous does the pulse become. If, on the other hand, the capillaries are dilated, the effect is the same; for, in either case, the collapse of the artery is accelerated, and the period of its expansion abbreviated.

Having thus endeavoured to establish certain principles for the guidance of the clinical observer in the interpretation of sphygmographic tracings, I proceed to illustrate these principles by reference to

cases; but, before doing so, would guard myself, as well as warn those who are looking hopefully to the sphygmograph as a new aid in clinical research, against the error to which our practical bias is so apt to lead us. The tendency to force theories to practical ends before they have had time to be established, is one of which we have many instances in the history of medicine. Let us remember that no theory ought to be applied to the issues of practical life, until it has been subjected to scientific criticism, and received as a scientific truth. Let us, therefore, avoid being in too great a hurry to introduce the sphygmograph into the consulting-room; for if, with so imperfect a knowledge as we at present possess of first principles, we endeavour to use those principles in diagnosis, we shall not only discredit ourselves, but the method by which we profess to be guided.

The practical information which will be eventually obtained by the use of the sphygmograph will clearly not differ in nature from that which we now obtain from the exploration of the pulse in the ordinary way. It is not likely that, with the one exception of certain aneurisms, the instrument will ever be of much use in the discovery of organic lesions. Its value will, in my judgment, consist in the exactitude and precision which it will impart to the notions we at present possess as to the practical significance of the various forms of pulse. Every one will admit

that our knowledge of the pulse is in this sense vague, that it is difficult to record it in words. In another, and more important, sense, it is exact; for he who has acquired the habit of discriminating pulses instinctively—a habit which, I apprehend, is a great deal more rare now than it used to be—instinctively learns valuable truths from it, which he can apply to practice. Yet how difficult—how impossible—is it for the skilled physician to impart this knowledge to his less-experienced junior! How difficult to describe to the student what you feel when you place your finger on the wrist! How infinitely more easy, for example, is it to describe a *bruit* or a friction-sound, and to make either of them clearly understood, than a pulse; how inconsistent are the classifications of pulses found in text-books, and how various the opinions given by different physicians as to the same pulse!

That the use of the sphygmograph will tend to clear up all this uncertainty, I can entertain no doubt; for, whatever else may be questioned, it cannot be denied that it is an impartial and consistent witness. Notwithstanding the infinite variety of form observed in different persons, the tracing of the pulse of the same person is the same at all times, so long as he is in health. The normal succession of events is so constant, that every departure from it affords evidence of disease.

The conditions of the circulation which manifest themselves in the pulse, independently of organic changes in the heart or arteries, are, as has already been stated in the introduction, those which relate principally, either to the mode in which the left ventricle contracts—whether sudden or gradual, or to the relative quantities of blood contained in the arteries and veins. I will endeavour first to give examples of those forms of pulse in which the arterial pressure is defective. Be it remembered, that it is the function of the heart to fill the arteries and empty the veins; that, in proportion as the energy of the heart diminishes in disease, the veins become fuller and the arteries emptier; that all modes of death, excepting death by syncope or shock, consist in gradual depletion of the arteries; and that in almost all fatal disease, the signs which indicate the tendency to a fatal termination are those which imply sinking of arterial pressure and increased distension of the veins. In health, the arterial pressure is such as to balance a column of mercury six or seven inches in height; but, even with a pressure of one or two inches, the circulation may be continued for a long time. It is only when it sinks to zero that systemic life is extinct.

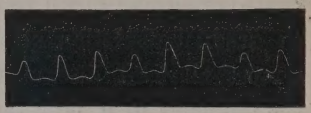
In those diseases in which the powers of life ebb gradually away, the pulse assumes, during the last stages, a form of which examples are given in the

figs. 1 and 2. This form denotes that the exhausted heart is no longer able to maintain the normal difference between the arterial and venous pressures,* so that, by the increase of the one and diminution of the other, they approach each other more and more closely. In both of these cases, the volume of the heart was known during life to be greatly increased, while in one of them (No. 1) a loud and harsh systolic murmur was heard over the præcordia, which was erroneously supposed to indicate disease of the mitral valve. In both, the superficial cervical veins were distended, and pulsated visibly. After death, it was found that the left ventricles were

Fig. 1.

Pulse of exhaustion. J. E., aged 38.

Fig. 2.



Pulse of exhaustion with excitement. M. O., aged 68.

hypertrophied and the right cavities dilated, and that the abdominal organs exhibited those evidences of venous congestion which are usually associated with so-called obstructive disease of the heart. A pulse of this kind is truly described as weak or failing. It often seems to vibrate under the finger, a character which is due to its double beat. The

* It is usually estimated that the pressure on the arteries in health is about twenty times that which exists in the veins.

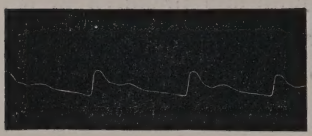
primary expansion is so feeble, that it is not transmitted so far as to the wrist, but can be readily demonstrated by applying the sphygmograph to the femoral artery at the groin.

Sometimes the second beat is so well marked as almost to equal the first in intensity. The explanation of this fact, which depends on the elastic resistance of the arteries, may be thus stated. So long as the vigour of the cardiac systole remains unchanged, the greater the elastic resistance, the less is the disturbance of pressure-equilibrium produced by it. In other words, when the arteries are relatively tense—i.e. tense as compared with the force with which the heart contracts, the vascular distension at the moment of systole is equal throughout the whole system, and its subsidence is continuous and unbroken, as in the normal state of the circulation. If, on the other hand, the arterial resistance be feeble, the immediate increase of tension is not only slighter, but more partial—being much greater in those parts of the system which are nearest the heart than in those which are more distant. By following out these considerations, we arrive at the general principle, that the greater the inequality of tension produced at the moment of the contraction of the heart, between the peripheral arteries, and the great trunks, the stronger is the second beat as compared with the first. It will be understood that this explanation,

which is the same in substance as that which I gave more generally in the introduction, is applicable to every case of dicrotism.*

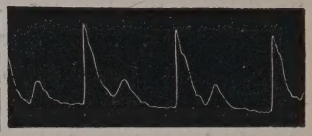
Fig. 3 is the pulse of a man of 25, convalescent from an attack of pneumonia. It is the pulse of mere arterial atony, such as is commonly met with in healthy persons after recovery from acute diseases. During his illness, the pulse of this patient had assumed a form analogous to fig. 4, but of much

Fig. 3.



Pulse of convalescence from acute disease. S. J., aged 25.

Fig. 4.



Pulse of nervous excitement. W. H., aged 18.

greater frequency. The pyrexial sharpness with which the heart contracted, showed itself in an excessively sudden increase of arterial tension, which, in consequence of the great yieldingness of the arteries, was of short duration. The closure of the aortic valve was immediately followed by a diastolic wave of great intensity; with reference to which it was interesting to notice that, in this, as in all other instances in which I have had occasion to make the observation, the pyrexial acceleration of the pulse

* For a more full exposition of the phenomenon of dicrotism, see p. 22.

was not attended with any change in the duration of the systolic period—that is to say, the heart beat more frequently, not because its movements were more quickly performed, but because it took a shorter time to repose between them. The time intervening between the first and second beat, the shock and the counter-shock, remained the same, although at one time the pulse was 70, at the other 110. Whether the emptiness of the arteries in convalescence depends on diminution of the mass of the circulating blood (spanæmia), or on relaxation of the capillary arteries, either of which would serve, on purely physical principles, to explain it, is uncertain. I am inclined to attribute most importance to the latter.

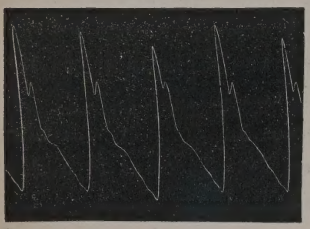
I refer to pulse No. 4 principally for the purpose of showing how completely the sphygmographic form may be modified by merely functional, that is to say nervous, disorder; and particularly how impossible it is to discriminate between the effects of mere nervous excitement of the heart and that which accompanies pyrexia. It is the pulse of a young man aged 18, who, in the absence of either signs or symptoms of phthisis, presented himself at the hospital, believing himself to be 'in decline.' So far as relates to its form, it can hardly be distinguished from the more frequent dicrotous pulse of mild pyrexia, which is represented on Pl. I. fig. 3. In both cases, if the view which I suggested in the

introduction be correct, the *suddenness* of the contraction of the heart indicates its spinal character, although the part which the nervous centres take may be different.

Fig. 5 represents the pulse of a middle-aged man recently under the care of one of my colleagues at Brompton, who had acute rheumatism eight years ago, and several times since. When the tracing was taken, he was suffering from orthopnœa and præcordial pain. The impulse of the heart was to the left of the mammary line, and occupied a space as large as the palm of one's hand. A loud diastolic *bruit* was heard at the fourth cartilage, and a systolic bellows-sound over the aorta. Posteriorly there was dulness at both bases, and abundant subcrepitant *râles* were heard in inspiration. There could, therefore, be no doubt as to the nature of the case, which soon terminated fatally. After death, it was found that the aortic valve was so altered that the most copious regurgitation of blood must have taken place during each diastolic period. In this case, the pulse exhibits characters which, I believe, are not met with excepting in connection with aortic incompetence. These do not consist, as is often supposed, in the unusual verticality of the expansion; for, as has been already seen, this peculiarity may be produced by merely functional disorder. The distinctive peculiarity consists in the collapse. The tracing shows that

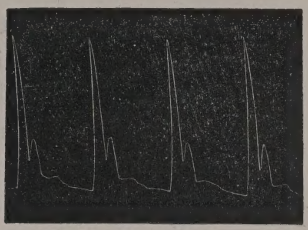
the artery becomes completely emptied during the interval between each beat and its successor; so that the diastolic expansion is no longer indicated. The explanation is simple. Immediately after the heart has ceased to contract, the blood injected into the aorta rushes back into the relaxed ventricle; so that, although the arterial equilibrium is for a moment disturbed, it is almost immediately re-established, the excess of pressure in the great arteries being at once

FIG. 5.



Pulse of Aortic regurgitation. H. R., aged 36.

FIG. 6.



The same. H. K., aged 38.

relieved. In other words, the elastic force, which is naturally expended in producing what is called the diastolic expansion, is wasted in regurgitation.

The next case (fig. 6) is that of a man of similar age, who in like manner had rheumatism about fifteen years ago, and several times since, but did not suffer from disease of the heart until his last attack in August 1866. The præcordial dulness in this case did not extend beyond the mammary line.

The impulse was diffused and expansive, and ended in a sudden retraction of the wall of the chest, easily felt by the hand. A diastolic *bruit* was heard at the fifth left cartilage, and a systolic *bruit* in the arteries, just as in the previous case. The tracing indicates that the primary expansion of the artery was sudden and excessively violent; and the collapse so complete, that the second beat is only slightly indicated. In such a pulse, one might imagine that the artery was entirely empty during the pause of the heart, but I have found that, by weakening the spring of the sphymograph, I could obtain tracings in which both the second event, of which the diagram gives no indication, and the diastolic collapse, are expressed.*

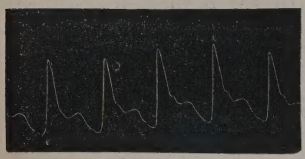
The striking peculiarities of form which the sphygmographic tracing exhibits in cases of aortic regurgitation, have afforded to some writers a ground for regarding the sphygmograph as an instrument of diagnosis in heart disease. The truth is, that all such cases can be just as satisfactorily made out by the ordinary methods of examination. In these two patients, for example, there was no manner of doubt as to the nature of the lesion. Indeed, the character

* This patient died a few days ago (September 1867). The aortic valve was found to be entirely incompetent, its curtain being shrivelled, thickened, and encrusted with vegetation. The mitral orifice was somewhat narrowed and the valve thickened, but not incapable of performing its functions. The right cavities of the heart were much dilated.

of the pulse was so obvious that, with the finger on the wrist, one might have made a diagnosis with one's eyes shut.

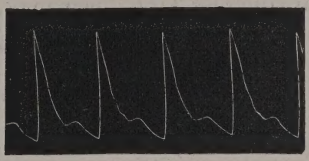
In the more numerous instances in which the same peculiarities present themselves in a less marked degree, the presumption they afford in favour of aortic regurgitation falls far short of evidence of its existence. To illustrate this, I refer to the two tracings, figs. 7 and 8. One (fig. 7), is the pulse of a man, aged 28, who was admitted into hospital with motor paralysis of the right arm and right side

Fig. 7.



J. S., aged 28. Mitral valvular disease.

Fig. 8.



J. B., aged 25. Aortic valvular disease. The two forms are essentially the same.

of the face, which had come on a few days before, somewhat gradually. With this exception, and that of slight metæsthesia of the affected arm, there was no disorder referable to the nervous system. The paralysis rapidly diminished, and the patient regained health and appetite. The tracing was taken three weeks after the seizure, when, on examination of the chest, I found that there was a bellows murmur, lasting during the whole systolic

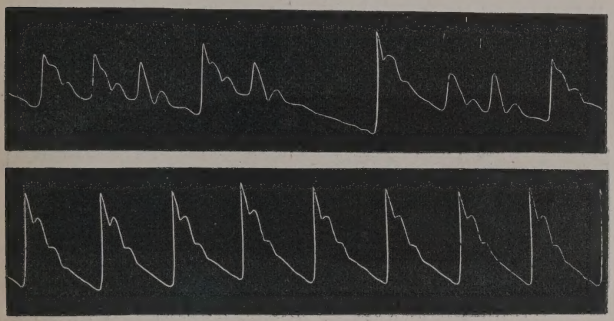
period at the apex, but no indication of aortic regurgitation. Let us compare with this the pulse of another patient (fig. 8), in whom the physical signs were so exactly similar to those already detailed with reference to fig. 6, that I need not occupy space in describing them. It is, in fact, so plain a case of aortic regurgitation, that the merest tyro in auscultation could not mistake it. The comparison of figs. 7 and 8 illustrates how completely the organic is simulated by the functional disorder of the heart.

To the question, What is the nature of this functional disorder, I venture to hope that the principles laid down may, in some measure, afford an answer. The condition of the heart which simulates aortic regurgitation, is that in which its contraction is smart, or even violent, but soon over. The bounding and collapsing pulse may, in fact, arise from any condition in which the heart contracts violently or suddenly, whatever be the arterial tension. If the arteries are tense, the pulse has the character understood by the term wiry; if they are relaxed, it is long and soft, and usually thrilling.

It is well known, that persons of gouty constitution are liable to be affected with irregularity of pulse quite independently of valvular lesion. In this affection, the irregularity is one of rhythm. During the period of inspiration, when, as I have elsewhere

shown, the frequency of the contractions of the heart is increased, the pulse becomes dicrotic, its form being entirely different from that which it assumes during the respiratory pause, when it is relatively retarded. During the respiratory pause, on the other hand, the contractions of the heart are less frequent; the diastolic intervals are longer, so that

FIG. 9.



T. R., aged 57. Irregular pulse of gout. In this patient, during the attack, the pulse becomes regular, as seen in the lower line. At other times, the largest sweeps of the lever occur during the respiratory pause; the smallest during and after inspiration.

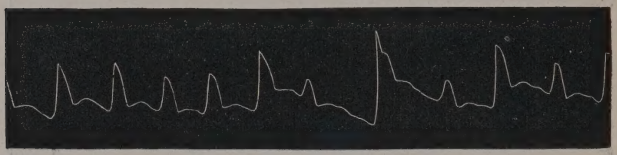
the heart has time to fill completely before it contracts. Hence, the quantity of blood delivered into the aorta is much larger in proportion to the quantity which can be transmitted by the capillaries. The duration of the ventricular systole is greater, and the arteries remain a much longer time distended. The pulse is no longer dicrotic.

Thus, in one and the same individual, you have

dicrotism during inspiration—absence of dicrotism during the respiratory pause; the only difference in the state of the circulation being that, in the one case, the diastolic pause is shortened, and, consequently, the ventricle contracts upon an insufficient supply of blood; whereas, in the other, its expansion is complete and its systole effectual.

Let us refer to another case. Fig. 10 shows a pulse often seen in rheumatic valvular disease, with large mitral regurgitation. The heart acts very irregularly. At times, the ventricle contracts

Fig. 10.



E. H., aged 58. Irregular pulse of mitral valvular disease.

effectually; but, at other times, the systolic expansion of the arteries is imperfect and abbreviated, in consequence of which the mean arterial tension declines. Whenever this is the case, the pulse assumes a form which is as distinctly dicrotic as that of typhus; so much so, indeed, that its double character can be readily recognised by the finger.*

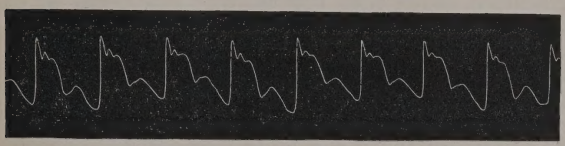
I have now said all that I have time to say of those forms of pulse which exhibit arterial atony.

* See Pl. I. fig. 2.

We have still to consider those which represent the opposite condition, that of excessive resistance.

Increased arterial resistance may be connected with a variety of causes. Thus, it may arise either from a contracted state of the capillaries, from diminished elasticity of the arteries, or from narrow-

FIG. 11.



G. M., aged 40. Pulse of aortic obstruction, with hypertrophy of the left ventricle.

ing of the aortic valve. Figs. 11, 12, and 13, serve to illustrate these points. An example of the most simple case of resistance to the contraction of the left ventricle—viz. that which occurs in aortic stenosis—is given in fig. 11. This is the pulse of a patient who died a few days ago in the hospital. The heart, with the adherent pericardium, weighs thirty ounces. The left ventricle is both thickened and dilated, and the aortic valve so deformed and beset with vegetations, that the orifice would scarcely admit the tip of the index finger. The other valves are healthy. All the other morbid appearances found were distinctly referable to the cardiac lesion as their cause. Thus, the kidneys, although enlarged and congested, are healthy. The tracing

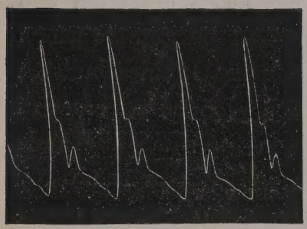
scarcely needs explanation; its characters are seen to be just the opposite of those we have been hitherto considering. The second event, which, in the pulse of aortic regurgitation, and indeed in all atonic pulses, is either entirely suppressed or difficult to distinguish, is here extremely well marked; and the form of that part of the curve pulse which represents it shows that the systolic expansion of the artery is of nearly equal duration with the diastolic interval.

The patient whose pulse is represented in fig. 12 was complaining, at the time when the observation was made, of dyspnœa on the slightest exertion, and præcordial pain. He was subject to nocturnal paroxysms of cardiac distress, which, he stated, always came on with pain at the scrobiculus, followed by palpitation and nausea, with violent respiratory efforts. On examining his chest, I found that the cardiac dulness extended from the sternum to the mammary line, and that the præcordial impulse was diffused and expansive. The systolic sound was prolonged, but no abnormal murmur could be made out. He died several months afterwards, when it was found that the heart weighed twenty ounces, and that the left ventricle was both hypertrophied and dilated, without valvular disease.

Fig. 13 is the pulse of a patient, aged 58, whose physical signs were very similar. The cardiac dul-

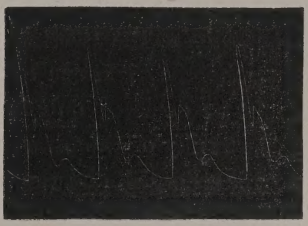
ness was not, however, so extensive, nor were the symptoms referable to the heart affection so distressing as in the last case. Both of these patients had bounding, visible pulses, the arteries knocking against the finger like a hammer; and in both, after death, it was found that the kidneys were much contracted, that their capsules were thickened and

FIG. 12.



W. S., aged 59. Hypertrophy of the left ventricle without valvular disease.

FIG. 13.



R. B., aged 58. The same.

adherent, that their surfaces were coarsely granular and studded with numerous cysts, and their cortices atrophied. The tracings, therefore, represent that condition of the heart which accompanies advanced renal degeneration, and appears to be its inevitable result. The differences between the two are not important. In the one case (fig. 12), the evidence of diminished elasticity is less marked than in the other.

I regret that time will not permit me to give more numerous examples. Those already placed before

you will, I trust, serve to illustrate the principle on which the discrimination of increased arterial resistance rests; viz. the exaggeration of the systolic distension, as compared with the diastolic. The state of the circulatory apparatus which these conditions imply, although it cannot be said to constitute a disease, is something more than a mere symptom. So strongly am I convinced of its value as an element of diseased action, that I regard its detection and estimation as the most important purpose to which the sphygmograph can be applied. If this condition could be detected only in well pronounced cases of hypertrophy of the heart when the præcordial dulness is enlarged, and the patient perhaps is already suffering from well marked signs of disordered circulation, it would be of little value. But if we could establish as a fact what appears more and more probable, that increased arterial resistance may not only exist independently of any other sign of organic disease, but may be detected with the aid of the sphygmograph, the practical value of the result would be unquestionable. With reference to it, I would, in conclusion, submit to you a consideration which appears to me to be interesting. Anatomical researches lead us to believe it probable that the earliest beginnings of what may be called degenerative disease consist in such structural alterations of the minutest arteries, as, by rendering them less

pervious to the circulating blood, must inevitably lead to increased arterial resistance estimable by the sphygmograph. If I am right in believing that this may be so measured—if, in other words, we can ascertain, by the examination of the pulse, that the heart is overtaxed long before any change can be detected by auscultation or percussion, it is obvious that we have made a step forward of practical utility.

In conclusion, let me state that it is with these considerations in view that I have elsewhere expressed the opinion, that the sphygmograph is likely to be of greater use as an aid in forming an opinion as to the probable duration of life, than in any other department of medical practice. There are many persons in whom, in the absence of any other trace of ailment, the pulse-curve indicates that the arterial resistance is excessive. The question is, Are such persons sound? I think not; but, as yet, we know too little to speak dogmatically.

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